

# Type A Aortic Dissection Presenting with Inferior ST-Elevation Myocardial Infarction

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Type A aortic dissection with concurrent ST-elevation myocardial infarction (STEMI) is relatively rare. However, it can be potentially fatal and easily misdiagnosed as STEMI alone. Misdiagnosis will lead to inappropriate administration of anticoagulant and thrombolytic therapy and delayed surgical repair of the aorta. In patients with STEMI, short reperfusion time is associated with improved survival, and minimizing the door-to-balloon time is the goal of therapy worldwide. However, signs critical for differential diagnosis may be overlooked in the rush to primary percutaneous coronary intervention. When a patient is encountered who presents with chest pain and ST elevation on electrocardiogram, STEMI should not be the only diagnosis considered. By using bedside available information, detailed history taking and focused physical examination, it is possible to avoid a mistaken diagnosis. Here we report a case of Stanford type A aortic dissection with STEMI that was initially misdiagnosed as sole acute inferior wall myocardial infarction. Patient mortality may have resulted from delayed diagnosis and surgical treatment.

**Key Words:** Acute myocardial infarction • Aortic dissection

## INTRODUCTION

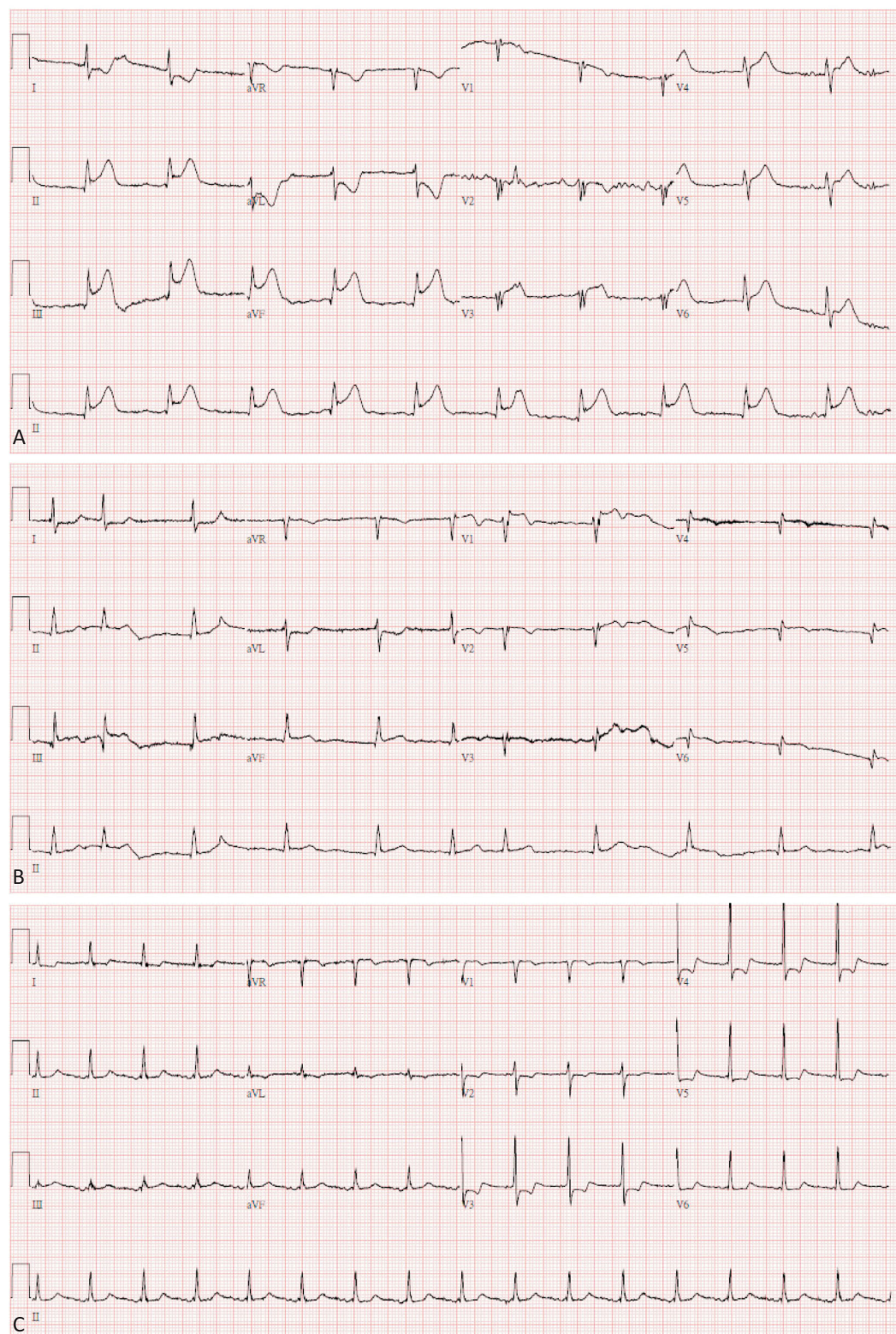
Aortic dissection can present with a wide range of manifestations, and its diagnosis can be challenging for primary care emergency physicians.<sup>1</sup> Approximately one-fourth of patients with acute aortic syndrome present with an electrocardiographic pattern resembling that of acute coronary syndrome. Patients with an electrocardiogram (ECG) suggestive of myocardial ischemia are easily misdiagnosed,<sup>2</sup> and the possibility of misdiagnosis is even higher in patients with concurrent ST-elevation myocardial infarction (STEMI).<sup>3</sup> Here we present a case with Stanford type A aortic dissection

and STEMI who failed to survive because he was initially misdiagnosed, leading to delayed diagnosis and surgical treatment.

## CASE REPORT

A 70-year-old man with a history of uncontrolled hypertension presented to our emergency department with acute onset of chest pain. The pain radiated to the patient's back and was associated with cold sweating. The ECG showed ST elevation in the inferior leads with reciprocal ST depression in leads I and aVL (Figure 1A). Hypotension was noted and treated with a vasopressor. Acute STEMI of the inferior wall with shock was impressed. A chest radiograph revealed mediastinal widening, a prominent aortic knob, and left pleural effusion (Figure 2A). Approximately 5 min later, significant resolution of ST segment elevation was observed on a right-sided ECG (Figure 1B). Hypotension persisted and the patient was sent for primary percutaneous coronary

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**Figure 1.** (A) An initial ECG showing ST elevation in the inferior leads with reciprocal ST depression in leads I and aVL. (B) Resolution of ST elevation on a right-sided electrocardiogram. (C) New ST depression in the anterior leads after primary percutaneous coronary intervention.



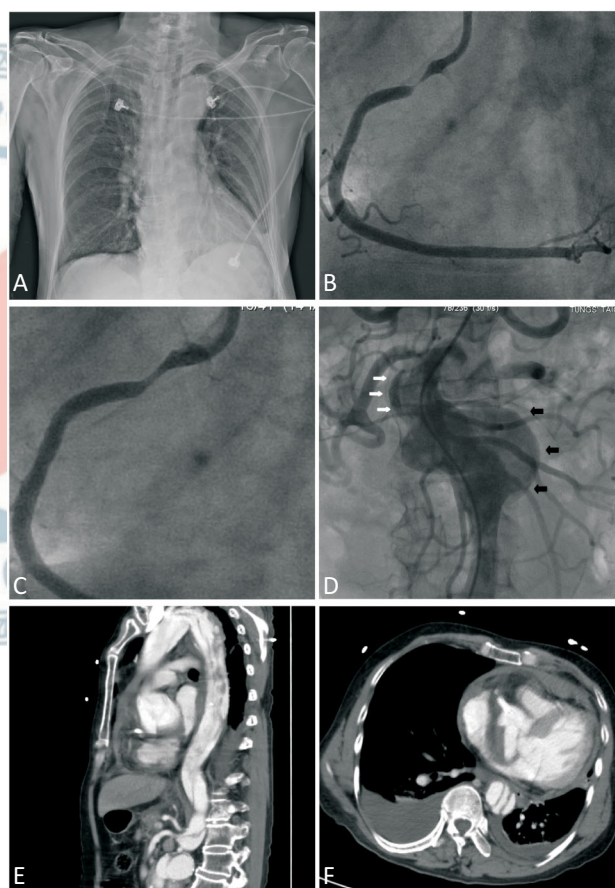
intervention (PCI). Coronary angiography revealed a lesion causing 50% stenosis in the middle left anterior descending artery and dynamic stenosis of the proximal right coronary artery (RCA) (Figure 2B). The culprit lesion in the proximal RCA was quite different from those responsible for most cases of STEMI. The luminal surface was relatively smooth, and there was no dissection plaque or thrombus. The RCA was almost totally occluded in the systolic phase, whereas flow improved in the diastolic phase (Figure 2C). The stenosis could not be relieved by vasodilator injection or angioplasty. A stent was inserted into the proximal RCA and Thrombolysis in Myocardial Infarction 3 grade flow was established. The door-to-balloon time was 79 min. Resistance was encountered while pushing the wire up the abdominal aorta during the procedure; therefore, abdominal aortography was performed after PCI. Aortography revealed a large abdominal aortic aneurysm (Figure 2D, black arrow), which explained the resistance encountered.

After PCI, the observed shock worsened. A follow-up ECG revealed complete resolution of ST elevation in the inferior leads; however, new ST depression had appeared in the anterior leads, suggesting anterior ischemia (Figure 1C). Heart echocardiography revealed hypokinesis of the inferior wall with normal anterior wall motion. In addition, a dilated aortic root with mild to moderate aortic regurgitation and mild pericardial effusion was also noted. Clinical information was reviewed again. A history of chest pain and hypertension, mediastinal widening and a prominent aortic knob on chest radiograph, an unusual coronary lesion and echocardiographic findings of aortic regurgitation and pericardial effusion all suggested aortic dissection. A dissection plane across the abdominal aneurysm was noted on the abdominal aortogram after careful review (Figure 2D, white arrow). Enhanced chest computed tomography (CT) showed a dissection plane extending from the aortic root to the left common iliac artery and across the abdominal aneurysm (Figure 2E). The false lumen perforated into the region of the aortic root, with formation of a periaortic hematoma that compressed the stented proximal RCA (Figure 2F). A final diagnosis of Stanford type A aortic dissection with RCA involvement and resultant acute inferior myocardial infarction was made. The time from arrival at the emergency de-

partment to definitive diagnosis was 16 h. The patient was transferred for emergency surgery, but he could not be weaned from cardiopulmonary bypass and died in the operating room.

## DISCUSSION

Acute myocardial infarction due to extension of an acute Stanford type A aortic dissection is a rare but devastating event, occurring in approximately 3% patients with aortic dissection.<sup>4</sup> The RCA is involved in most patients with concurrent infarction. Differentiating acute Stanford A aortic dissection with coronary mal-



**Figure 2.** (A) Mediastinal widening, abnormal aortic contour, and left pleural effusion on a chest radiograph. (B) Near total occlusion of the proximal RCA in the systolic phase. (C) Improved flow in the diastolic phase. (D) Aortography reveals an abdominal aneurysm (black arrow) with a dissection plane (white arrow). (E) The dissection plane extends across the abdominal aneurysm in the sagittal plane. (F) Perforation of the false lumen with periaortic hematoma formation and resultant compression of the stented proximal RCA.

perfusion from true acute myocardial infarction is challenging for emergency physicians, and misdiagnosis is very common in patients with ST segment elevation on initial ECGs.<sup>3,5</sup> Inappropriate treatment with antiplatelet, antithrombin, and thrombolytic agents can cause catastrophic bleeding.

In patients with STEMI, timely reperfusion improves the clinical outcome. The guidelines for STEMI recommended a door-to-balloon time of < 90 min and most medical personnel are under constant pressure to achieve this goal. The guidelines also contained the following recommendations to facilitate the differentiation of STEMI from aortic dissection:<sup>6</sup> "Evaluation of the patient's complaints should focus on chest discomfort, associated symptoms, sex and age-related differences in presentation, hypertension, diabetes mellitus, and the possibility of aortic dissection", "Patients with STEMI should have a portable chest X-ray, but this should not delay implementation of reperfusion therapy (unless a potential contraindication is suspected, such as aortic dissection)", and "Imaging studies such as a high-quality portable chest X-ray, transthoracic and/or transesophageal echocardiography, and contrast chest CT scan or magnetic resonance imaging scan should be used for differentiating STEMI from aortic dissection in patients in whom this distinction is initially unclear". However, under pressure to achieve short reperfusion times, important clinical clues for differential diagnosis may be overlooked.

Aortic dissection has been frequently misdiagnosed as acute coronary syndrome. Acute coronary syndrome has the diagnostic advantages of the ECG and cardiac enzymes testing, allowing risk stratification and emergent treatment. Aortic dissection has no equivalent diagnostic tests that are rapidly available. The International Registry of Acute Aortic Dissection Substudy on Biomarkers (IRAD-Bio) study evaluated the diagnostic performance of D-dimer testing in a study population of patients with suspected aortic dissection.<sup>7</sup> At the cutoff level of 500 ng/mL versus control, sensitivity and specificity were 96.6% and 46.6%, respectively. When the cutoff level was increased to 1600 ng/mL, D-dimer may be a useful tool in differential diagnosis among aortic dissection and myocardial infarction, angina or other ischemia heart diseases within the first 6 h. D-dimer, although imperfect due to its low specificity, is currently

the only commercially available test that can be used for the biochemical diagnosis of aortic dissection.

In the absence of a rapid, accurate, and readily available diagnostic test, the current diagnosis of aortic dissection requires comprehensive interpretation of available information. In 2010, the American Heart Association released guidelines for the diagnosis and management of patients with thoracic aortic disease, which introduced a risk assessment tool focusing on specific high-risk predisposing conditions, pain features, and physical examination findings. This aortic dissection detection risk score is highly sensitive (95.7%) for the detection of acute aortic dissection when applied to the patients diagnosed with aortic dissection in the IRAD database.<sup>8</sup> The results from this study suggested that the aortic dissection detection risk score, with the use of only information that is available at bedside, offers adequate sensitivity to capture the vast majority of patients presenting with acute aortic dissection.

In our patient, there were several indications of aortic dissection. First, detailed characterization of chest pain in the emergency department may have provided clues to aortic dissection. Typically, aortic dissection presents with severe tearing chest pain that instantly reaches its maximal intensity. The qualitative nature of the patient's chest pain was not clarified. Second, the patient had a history of uncontrolled hypertension, and chest radiography had revealed mediastinal widening, an abnormal aortic contour, and left pleural effusion, which should have prompted further imaging studies. Third, a murmur from aortic regurgitation or pulse deficit could have been detected on physical examination. Fourth, resistance encountered during catheterization is a sign of possible structural damage to the aorta or its branches. Although an abdominal aortogram was obtained, a dissection plane was overlooked. Fifth, the culprit vessel was not completely occluded and the lesion characteristics were quite different from those of conventional STEMI. The significant difference between central (aortic root) and peripheral (right femoral artery) blood pressure (around 50 mmHg) was also ignored during PCI.

In emergency departments, the number of patients treated for acute coronary syndrome is greater than that of patients treated for aortic dissection. Differentiating acute coronary syndrome from type A aortic dis-

section with concomitant coronary malperfusion is challenging, because of demands for minimal door-to-balloon time. Our findings from this case suggested that acute aortic dissection should always be considered as a differential diagnosis in patients presenting with symptoms suggesting acute coronary syndrome. If there is a high index of suspicion, it is wise to consider further imaging studies as the recommendation of the above-mentioned guidelines. Despite the importance of a short reperfusion time, achieving this goal should not compromise diagnostic accuracy.

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